ILLUSTRATED DICTIONARY OF IMMUNOLOGY

SECOND EDITION

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C

anti-CALLA monoclonal antibodies and complement. It is a pre-B lymphoblast marker that is the most frequent type of cell in childhood acute lymphocytic leukemia (ALL). The presence of Ia antigen with CALLA portends a favorable prognosis. CALLA may also be positive in Burkitt's lymphoma, B cell lymphomas, and 40% of T cell lymphoblastic lymphomas. All blasts are usually positive, not only for CALLA and the Ia antigen, but also for TdT.

common leukocyte antigen (LCA) (CD45)

An antigen shared in common by both T and B lymphocytes and expressed, to a lesser degree, by histiocytes and plasma cells. By immunoperoxidase staining, it can be demonstrated in sections of paraffin-embedded tissues containing these cell types. Thus, it is a valuable marker to distinguish lymphoreticular neoplasms from carcinomas and sarcomas.

common lymphoid progenitors

Stem cells from which all lymphocytes are derived. Pluripotent hematopoietic stem cells give rise to these progenitors.

common variable antibody deficiency

Refer to common variable immunodeficiency (CVID).

common variable immunodeficiency (CVID)

Common variable immunodeficiency is a relatively common congenital or acquired immunodeficiency that may be either familial or sporadic. The familial form may have a variable mode of inheritance. Hypogammaglobulinemia is common to all of these patients and usually affects all classes of immunoglobulin, but in some cases only IgG is affected. The World Health Organization (WHO) classifies three forms of the disorder: (1) an intrinsic B lymphocyte defect, (2) a disorder of T lymphocyte regulation that includes deficient T helper lymphocytes or activated T suppressor lymphocytes, and (3) autoantibodies against T and B lymphocytes. The majority of patients have an intrinsic B cell defect with normal numbers of B cells in the circulation that can identify antigens and proliferate but cannot differentiate into plasma cells. The ability of B cells to proliferate when stimulated by antigen is evidenced by hyperplasia of B cell regions of lymph nodes, spleen, and other lymphoid tissues. Yet, differentiation of B cells into plasma cells is blocked. The deficiency of antibody that results leads to recurrent bacterial infections, as well as intestinal infestation by *Giardia lamblia,* which produces a syndrome that resembles sprue. Noncaseating granulomas occur in many organs. There is an increased incidence of autoimmune diseases, such as pernicious anemia, rheumatoid arthritis, and hemolytic anemia. Lymphomas also occur in these immunologically deficient individuals.

competency, immunologic

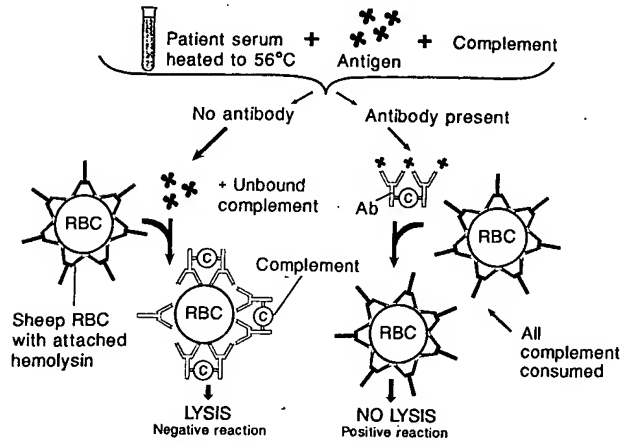
The capacity of an animal's immune system to generate a response to an immunogen.

competitive binding assays

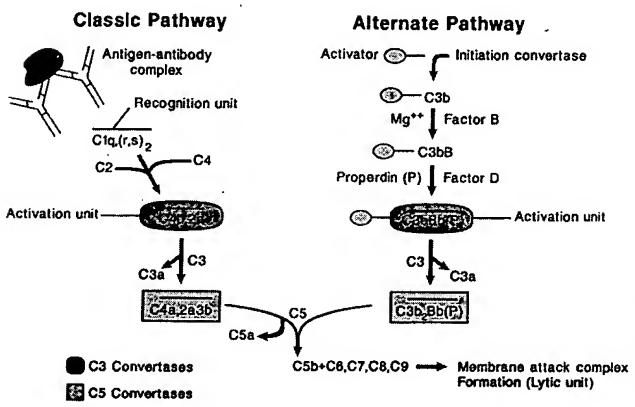
Serological tests in which unknowns are detected and quantified by their ability to inhibit binding of a labeled known ligand to a specific antibody. Also termed competitive inhibition assay.

competitive inhibition assay

A test in which antigens or antibodies are assayed by binding of a known anitbody or antigen to a known amount of labeled antibody or antigen. Known or unknown sources of antibody or antigen are then used as competitive inhibitors. Also termed competitive inhibition assay.



Complement split products in an inflammatory response.



Complement.

complement (C)

A system of 20 soluble plasma and other body fluid proteins together with cellular receptors for many of them and regulatory proteins found on blood and other tissue cells. These proteins play a critical role in aiding phagocytosis of immune complexes, which activate the complement system. These molecules and their fragments, resulting from the activation process, are significant in the regulation of cellular immune responsiveness. Once complement proteins identify and combine with target substance, serine proteases are activated, which ultimately leads to the assembly of C3 convertase, a protease on the surface of the target substance. The enzyme cleaves C3, yielding a C3b fragment that is bound to the target through a covalent linkage. C3b or C3bi bound to phagocytic cell surfaces become ligands for C3 receptors, as well as binding sites for C5. The union of C5b with C6, C7, C8, and C9 generates the membrane attack complex (MAC), which may associate with the lipid bilayer membrane of the cell to produce lysis, which is critical in resistance against certain species of bacteria. The complement

$CD4^+$ and $CD8^+$ T lymphocytes, together with TNF-α, IFN-γ, and IL-12, help to clear bacteria from the tissues, but antibody is also required in addition to cell-mediated immunity. The antibody may be directed against lipopolysaccharide (LPS) in animal infections with these microorganisms, and in humans antibodies to Vi antigen are believed to be significant. The diagnosis of typhoid fever is based on the detection of antibodies to O and H antigen and to Vi antigens in carriers. Immunization with killed *Salmonellae* does not induce cell-mediated immunity and confers less protection than immunization with live organisms that induce both cell-mediated and humoral immunity. Antibody alone is also not protective. The protective immunogen is probably LPS O antigen or Vi antigen in *S. typhi*, other protein antigens, or some combination of these. IgA provides partial immunity. Because killed microorganisms used in vaccines in the past do not induce appropriate cell-mediated immunity and are highly reactogenic, live vaccines of superior efficacy in experimental models are being developed.

salting out

Salt precipitation of serum proteins such as globulins.

salt precipitation

An earlier method to separate serum proteins based on the principle that globulins precipitate when the concentration of sodium sulfate or ammonium sulfate is less than the concentration at which albumin precipitates. Euglobulins precipitate at concentrations that are less than those at which pseudoglobulins precipitate. This method was largely replaced by chromatographic methods using Sephadex® beads and related techniques.

SAMS

Abbreviation for substrate adhesion molecules.

Sanarelli–Shwartzman reaction

Refer to Shwartzman reaction.

Sandoglobulin®

Refer to human immune globulin.

sandwich ELISA

A method in which surface-bound antibody traps a protein by binding to one of its epitopes. An enzyme-linked antibody specific for a different epitope on the protein surface is employed to detect the trapped protein. This is a highly specific assay.

sandwich immunoassay

A technique in which the analyte is bound to a solid phase, and a labeled reagent is subsequently bound immunochemically to the analyte.

"sandwich" methodology

Refer to sandwich technique.

sandwich technique

The identification of antibody or of antibody-synthesizing cells in tissue preparations in which antigen is placed in contact with the tissue section or smear, followed by the application of antibody labeled with a fluorochrome such as fluorescein isothiocyanate (FITC) that is specific for the antigen. This yields a product consisting of antibody layered on either side of an antigen, which accounts for the name "sandwich."

saponin

A glucoside used in the past for its adjuvant properties to enhance immune reactivity to certain vaccine constituents. It was considered to slow the release of immunogen from the site of injection and to induce B cells capable of forming antibody at the site of antigen deposition.

Open-lung biopsy showing sarcoidosis.

Open-lung biopsy showing sarcoidosis.

sarcoidosis

A systemic granulomatous disease that involves lymph nodes, lungs, eyes, and skin. A granulomatous hypersensitivity reaction resembles that of tuberculosis and fungus infections. Sarcoidosis has a higher incidence in African-Americans than in Caucasians and is prominent geographically in the southeastern United States. It is of unknown etiology. Immunologically, there is a decrease in circulating T cells. Decreased delayed-type hypersensitivity is manifested as anergy to common skin-test antigens. Increased antibody formation leads to polyclonal hypergammaglobulinemia. A marked cellular immune response is observed in local areas of disease activity. Tissue lesions consist of inflammatory cells and granulomas, composed of activated mononuclear phagocytes such as epithelioid cells, multinucleated giant cells, and macrophages. Activated T cells are present at the periphery of the granuloma. $CD4^+$ T cells appear to be the immunoregulatory cells governing granuloma formation. Mediators released from T cells nonspecifically stimulate B cells, resulting in the polyclonal hypergammaglobulinemia. The granulomas are typically noncaseating, distinguishing them from those produced in